

Prevalence of Cardiovascular Disease Risk Factors Among US Adults With Self-Reported Osteoarthritis: Data From the Third National Health and Nutrition Examination Survey

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Abstract

Objective: To estimate the prevalence of traditional risk factors for cardiovascular disease (CVD) among US adults with osteoarthritis (OA).

Methods: Using survey data from the Third National Health and Nutrition Examination Survey, we estimated the prevalence of selected CVD risk factors among a US OA and nonarthritic adult population. In additional analyses, we stratified the sample by gender and age (35-44, 45-64, and 65+ years) to further understand the CVD risk profile in an arthritic population and nonarthritic population. Relevant data on each survey participant's demographics, arthritis status, CVD risk factors, and sampling weights were obtained from the survey database.

Results: Of the 115.9 million US adults aged ≥ 35 years, 24.3 million (21%) have OA. Hypertension is prevalent in approximately 40% of OA patients; 20% of the patients smoke and 11% have diabetes. Prevalence of high total cholesterol is estimated to be 32%, while prevalence of low high-density lipoprotein cholesterol is estimated at 13%. Approximately 37% of OA patients are estimated to have renal impairment, but less than 1% suffer from renal failure.

Conclusion: National survey data suggest that, on average, US adults with OA have a high prevalence of cardiovascular risk factors. These findings highlight the need to consider patients' comorbidities when selecting the appropriate treatment options.

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million outpatient visits and three quarters of a million hospitalizations annually are attributable to arthritis and its treatment, with associated direct medical care costs of \$15 billion.^{1,2} Estimates of lost productivity and other indirect costs of arthritis have been estimated to be as high as \$50 billion annually.^{1,2} The clinical and economic burdens of arthritis in the United States are expected to increase as the general population ages; an estimated 60 million Americans (nearly 20% of the population) are projected to have arthritis by 2020, of whom approximately one fifth (or 12 million people) will experience meaningful activity limitation.^{1,3-5}

There is credible evidence that people with osteoarthritis (OA) and rheumatoid arthritis (RA) are at higher risk than the general population for several comorbid conditions, particularly cardiovascular disease (CVD).⁶⁻⁸ Moreover, there is an established body of research suggesting that age-adjusted mortality risk is higher among RA patients relative to the general population.⁷⁻¹⁷

The etiology of the association between arthritis and CVD is not fully understood. Various theories about the relationship have been put forth in recent studies, most of which are based on the premise that patients with arthritis are at advanced risk for development of CVD by virtue of their unfavorable risk factor profile. However, there is inherent difficulty in sorting out the relevant causes of CVD

Arthritis is a widely prevalent, disabling disease that places substantial demands on healthcare resources. It has been estimated that as many as 44

in arthritis patients as they differ from the general population in many aspects. Changes in body mass composition, changes in lipid profile associated with medication use (eg, glucocorticoids), and activity limitations resulting from chronic joint disease may all play a role in increased CVD risk.^{18,19} Some investigators believe that vascular inflammation associated with increased levels of thiol compounds and C-reactive protein, as well as peroxidization of low-density lipoprotein, may play a significant role in CVD pathogenesis in patients with arthritis.^{18,20} Medications taken for arthritis-related conditions have also been implicated for either directly or indirectly leading to atherosclerosis.^{18,21,22} The most commonly implicated drugs are glucocorticoids (chiefly, prednisone), which can increase serum lipids and glucose levels and induce hypertension.²³ Methotrexate, another commonly prescribed arthritis medication, has been shown to increase serum homocysteine levels.^{23,24}

Although national estimates of OA and RA prevalence have been reported,^{1,3,4} to the best of our knowledge the prevalence of CVD risk factors among such people has not been estimated to date. To this end, the government-sponsored database on the health status of the US population—the Third National Health and Nutrition Examination Survey (NHANES III)—was used to develop national estimates of the prevalence of selected cardiovascular risk factors among adult patients with self-reported OA and a nonarthritic US adult population.

...MATERIALS AND METHODS...

Data Source

We estimated the prevalence of selected CVD risk factors among US adults aged ≥ 35 years by diagnosis, gender, and age category (35-44, 45-64, and 65+ years) using survey data from NHANES III.²⁵

NHANES is one of the major programs in the series of health-related studies conducted by the National Center for Health Statistics, part of the US Centers for

Disease Control and Prevention, over the past 35 years. NHANES is designed to assess the health and nutritional status of adults and children in the United States through interviews and direct physical examinations. The survey is unique in that it combines a home interview with physical examinations and a variety of diagnostic and laboratory tests conducted in a mobile examination center. NHANES III, which was conducted from 1988 to 1994, included approximately 40 000 people aged ≥ 2 months selected from households in 81 counties across the 50 US states. Using a complex, stratified, multistage probability cluster sampling design (with oversampling of young children, older people, blacks, and Mexican Americans), the survey yields nationally representative information on the health and nutritional status of the civilian, non-institutionalized US population. Physical examinations and objective measures are employed when information cannot be furnished or is not available in a standardized manner through interviews or through records maintained by the health professionals who provide medical care to survey respondents.²⁵⁻²⁷

The 4 data files representing the major components of NHANES III are adult household, examination, laboratory, and dietary recall; more than 5000 data elements are collected. One section of the household adult questionnaire asks respondents to note whether a physician has told them that they have OA or RA, and when they were first told that they had the condition. Other sections of the questionnaire focus on diabetes, high blood pressure, CVD, musculoskeletal conditions, gallbladder disease, kidney conditions, respiratory and allergy conditions, vision and hearing, and dental care. Histories of smoking and chewing tobacco use are recorded on both the home adult questionnaire and examination questionnaire, while history of alcohol use is asked on the examination questionnaire. Other NHANES sections pertain to exercise, nutrition assessment, medicine/vitamin use, biochemistry values, and physical exam results. Biochemistry data collected con-

sist of hematologic tests, general biochemistry tests, urine tests, antibody tests, and diabetes testing profile. The physical exam consists of a physician's exam, dental examination, allergy skin test, audiometry, spirometry, bone densitometry, gallbladder ultrasonography, and fundus photography.^{25,26}

CVD risk factors examined in this study, as derived from the Household Adult Questionnaire (HAQ) and Laboratory Data File components of the NHANES III database, include systolic blood pressure (SBP) and diastolic blood pressure (DBP), total and high-density lipoprotein (HDL) cholesterol, physician-diagnosed diabetes mellitus, renal impairment or failure based on serum creatinine levels, and current cigarette smoking. Arthritis status was derived from the arthritis section of the HAQ as described above. Smoking status was derived from the question, "Do you smoke cigarettes now?" Diabetes mellitus status was derived from questions asking respondents whether a doctor had ever told them that they have diabetes. All other risk factor data were obtained from the NHANES III Laboratory Data File. Hypertension as a CVD risk factor was defined as SBP >140 mm Hg or DBP >90 mm Hg, as defined by current National Institutes of Health *Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure* (JNC VI) guidelines.²⁸ Renal impairment and failure were defined respectively as serum creatinine levels exceeding the upper limit and twice the upper limit of normal (ie, >1.5 mg/dL and >3 mg/dL, respectively), which reflects the methods of the Massachusetts General Hospital.²⁹

Statistical Analyses

Prevalence (stated as percentages) and associated 95% confidence intervals (CIs) were estimated for each CVD risk factor among an OA and a nonarthritic population. Gender- and age-stratified prevalence rates were also estimated for each population. SUDAAN[®] statistical analysis software (Research Triangle Institute,

Research Triangle Park, NC) in conjunction with Statistical Analysis System (SAS) Release 8.02 (SAS Institute, Cary, NC) were used for these analyses. SUDAAN is specifically designed for analysis of cluster-correlated data from surveys such as NHANES III that involve multi-stage sample designs. Robust variance estimates are generated that account for intracluster correlation, unequal weighting, stratification, and without-replacement sampling. To provide estimates that were representative of the US population, analyses of each data element incorporated sampling weights obtained from the NHANES III database. These weights account for the unequal probabilities of selection resulting from the cluster design, the planned oversampling of certain demographic subgroups, and nonresponse adjustment factors based on US Census Bureau data on age, gender, race, income, and geographic location of the US population.^{26,27} Since our investigation focused on interval estimation rather than on hypothesis testing, no tests of statistical significance were undertaken.

...RESULTS...

Prevalence Estimates

Osteoarthritis. Of the 115.9 million US adults aged ≥35 years, 24.3 million (21%) have OA (95% CI, 22.1 million-26.6 million) (Table 1). Nearly two thirds of these people are women. Prevalence rates of OA increase with age in both genders. However, the ratio of females to males with OA increases with advancing age, from 1.32:1 among people aged 35 to 44 years to 1.88:1 among people aged ≥65 years.

Table 1 also shows that the nonarthritic population is considerably younger than the OA population. Over 47% of OA patients are older than 65 years, compared with only 19% of those in the nonarthritic population. In addition, there were gender differences between the arthritis and nonarthritic populations; nearly 63% of the OA population was comprised of women, compared with only 49.9% of the general nonarthritic population.

Table 1. Estimated Numbers of US Adults Aged ≥ 35 Years With Osteoarthritis, by Gender and Age

Gender and Age (Years)	Osteoarthritis		General Population Without Arthritis	
	Percentage of People (95% CI)		Percentage of People (95% CI)	
All (n)	24 345 370	(22 110 212-26 580 528)	85 800 548	(78 999 986-92 601 110)
35-44	13.30	(11.79-14.56)	41.41	(40.18-42.47)
45-64	39.49	(39.26-39.68)	39.59	(39.74-39.46)
65+	47.21	(45.01-49.04)	18.99	(17.85-19.97)
Men (n)	9 015 680	(7 874 587-10 156 773)	42 986 882	(39 689 389-46 284 375)
35-44	15.51	(12.70-17.69)	40.13	(38.36-41.65)
45-64	40.23	(38.75-41.38)	41.17	(41.02-41.30)
65+	44.26	(42.54-45.59)	18.70	(17.63-19.62)
Women (n)	15 329 690	(13 824 691-16 834 689)	42 813 666	(38 878 456-46 748 876)
35-44	12.00	(9.99-13.66)	42.70	(41.43-43.77)
45-64	39.05	(37.58-40.26)	38.01	(37.44-38.48)
65+	48.95	(46.67-50.82)	19.29	(17.81-20.52)

CI indicates confidence interval.

Hypertension. Approximately 40% (95% CI, 35.3-45.5) of people with OA have Stage I-III hypertension as defined by the JNC VI guidelines (Table 2).²⁸ By comparison, only about 25% (95% CI, 23.0-27.6) of the general population without arthritis was estimated to have hypertension. Prevalence of hypertension is slightly higher among men than women, and, as epidemiologic data suggest, higher among people aged ≥ 65 years versus younger people.

Cigarette Smoking. Approximately 20% (95% CI, 17.6-23.1) of OA patients are current cigarette smokers (Table 2). The crude rates in this analysis are slightly lower than that for the general population without arthritis, wherein about 26% (95% CI, 23.6-28.4) are smokers.

Diabetes Mellitus. Approximately 11% (95% CI, 9.2-12.9) of people with OA have diabetes mellitus (Table 2). By comparison, only about 6% (95% CI, 5.6-7.3) of the general population without arthritis is diabetic. When stratifying by gender, this analysis suggests that female OA patients were more likely to have diabetes mellitus than a nonarthritic population. Prevalence of diabetes is slightly higher among

women than men, and, as epidemiologic data suggest, higher among older people.

Hypercholesterolemia. Approximately 32% (95% CI, 27.1-36.2) of people with OA have high total cholesterol levels (ie, ≥ 240 mg/dL) (Table 2). About 24% (95% CI, 21.4%-26.0%) of the general population without arthritis has high total cholesterol levels. Prevalence of high total cholesterol is slightly greater among women than men, and has a marked increase among people aged 45 years and older.

Low HDL Cholesterol. The prevalence of low HDL cholesterol (< 35 mg/dL) is similar, approximately 13% (95% CI, 10.8-16.1) in people with OA and 12% (95% CI, 10.4-13.2) in the general population without arthritis (Table 2). Prevalence of low HDL cholesterol is substantially higher among men than women, but there is little differentiation among the age categories.

Renal Impairment and Failure. Approximately 37% (95% CI, 31.6-41.5) of people with OA have renal impairment, manifested as serum creatinine levels exceeding the upper normal limit of 1.5 mg/dL (Table 2). Moreover, approximately 0.8% (95% CI, 0.4-1.3) of people with OA

have renal failure, defined as serum creatinine levels exceeding twice the upper normal limit (ie, ≥ 3.0 mg/dL). By comparison, it is estimated that only about 27% (95% CI, 23.8-30.3) of the general population without arthritis have renal impairment, and only 0.3% (95% CI, 0.2-0.4) have renal failure.

...DISCUSSION...

Patient-level examination data from the NHANES III have been used to estimate the prevalence of selected CVD risk factors among US adults with OA. Other studies assessing the prevalence of arthritis in the United States have been conducted, but the prevalence of traditional risk factors for CVD among arthritis patients has not been well quantified to date.

Estimates suggest that approximately 24.3 million US adults aged ≥ 35 years have OA, and that nearly two thirds of these people are women. These estimates are consistent with what has been reported elsewhere.³

Findings suggest that US adults with OA indeed may be at an increased risk of CVD relative to the nonarthritic population. For each of the risk factors examined, except cigarette smoking, point estimates of prevalence among OA patients exceeded those of the general population. While tests of statistical significance were not performed, it was observed that the difference in risk factor prevalence versus the general population is not statistically significant at the "conventional" $\alpha = 0.05$ level.

Our findings suggest that the prevalence of hypertension is significantly greater among OA patients versus patients without arthritis. Gabriel and colleagues⁶ found the prevalence of diabetes to be 5.0% among 441 OA patients. The estimates from this study at 11% are considerably higher. This difference could be related to a different population sampling in the 2 studies.

Reports on total cholesterol levels among patients with arthritis are scant

Table 2. Estimated Prevalence of CVD Risk Factors Among US Adults Aged ≥ 35 Years With and Without Osteoarthritis

Cardiovascular Disease Risk Factors, Stratified by Gender and Age (Years)	Prevalence, % (95% CI)			
	Osteoarthritis (n = 24 345 370)		General Population Without Arthritis (n = 115 861 005)	
Hypertension (Stage I-III, JNC VI Guidelines*)				
All	40%	(35.3-45.5)	25%	(23.0-27.6)
35-44	14%	(7.5-19.7)	11%	(9.3-13.1)
45-64	32%	(26.4-37.3)	28%	(24.6-31.5)
65+	55%	(46.8-63.5)	50%	(42.9-57.8)
Men	41%	(33.4-47.7)	28%	(25.1-31.0)
35-44	20%	(6.8-33.1)	14%	(11.1-17.7)
45-64	33%	(22.1-43.5)	32%	(27.4-36.7)
65+	55%	(45.5-64.2)	49%	(41.2-56.0)
Women	40%	(34.9-45.7)	23%	(19.8-25.4)
35-44	9%	(2.8-14.8)	8%	(6.1-10.3)
45-64	31%	(25.1-37.3)	24%	(20.2-27.2)
65+	55%	(45.7-64.8)	52%	(41.8-62.6)
Cigarette Smoking				
All	20%	(17.6-23.1)	26%	(23.6-28.4)
35-44	25%	(16.1-34.5)	31%	(27.2-34.8)
45-64	31%	(26.1-35.9)	26%	(22.6-29.3)
65+	10%	(8.1-12.2)	15%	(12.3-17.8)
Men	25%	(19.5-29.9)	30%	(26.9-33.2)
35-44	34%	(13.5-54.2)	36%	(30.7-42.3)
45-64	35%	(24.6-44.7)	29%	(24.5-34.2)
65+	12%	(8.2-16.6)	18%	(13.4-22.1)
Women	18%	(15.2-20.5)	22%	(19.1-24.7)
35-44	19%	(11.1-26.4)	26%	(21.4-30.4)
45-64	29%	(23.0-34.5)	22%	(18.2-26.2)
65+	9%	(6.4-11.5)	12%	(9.9-14.8)
Diabetes Mellitus				
All	11%	(9.2-12.9)	6%	(5.6-7.3)
35-44	4%	(0.7-7.2)	4%	(2.3-5.1)
45-64	11%	(7.3-13.8)	7%	(5.8-8.3)
65+	13%	(11.0-15.9)	11%	(9.0-12.8)
Men	10%	(7.3-12.3)	7%	(5.4-7.7)
35-44	0%	(-0.2-1.1)	3%	(1.0-5.3)
45-64	9%	(4.2-13.5)	8%	(6.0-10.0)
65+	14%	(9.8-18.1)	11%	(8.8-13.2)
Women	12%	(9.5-14.1)	6%	(5.0-7.5)
35-44	7%	(1.0-12.4)	4%	(2.6-6.1)
45-64	12%	(7.6-15.7)	6%	(4.4-7.7)
65+	13%	(10.3-16.1)	11%	(7.9-13.8)
High Total Cholesterol (≥240 mg/dL)				
All	32%	(27.1-36.2)	24%	(21.4-26.0)
35-44	22%	(11.6-32.5)	15%	(12.9-17.5)
45-64	34%	(27.8-39.3)	29%	(26.0-31.9)
65+	33%	(26.3-39.3)	31%	(25.9-36.7)
Men	23%	(17.7-28.5)	23%	(20.2-25.6)
35-44	22%	(10.5-33.9)	19%	(15.7-22.5)
45-64	26%	(18.6-33.4)	27%	(23.4-31.0)
65+	21%	(13.9-27.6)	21%	(17.1-25.7)
Women	37%	(30.9-42.7)	24%	(21.8-27.1)
35-44	22%	(8.9-35.0)	12%	(9.2-13.9)
45-64	38%	(29.9-46.5)	31%	(27.2-34.5)
65+	39%	(31.4-47.3)	41%	(32.8-49.2)

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*Systolic blood pressure >140 mm Hg; diastolic blood pressure >90 mm Hg (NIH Publication 98-4080, November 1997).

CVD indicates cardiovascular disease; JNC VI, *Sixth Report of the Joint National Committee on Prevention Detection, Evaluation, and Treatment of High Blood Pressure*.

Table 2. Estimated Prevalence of CVD Risk Factors Among US Adults Aged ≥35 Years With and Without Osteoarthritis (*Continued*)

Cardiovascular Disease Risk Factors, Stratified by Gender and Age (Years)	Osteoarthritis (n = 24 345 370)		General Population Without Arthritis (n = 115 861 005)	
Low HDL Cholesterol (<35 mg/dL)				
All	13%	(10.8-16.1)	12%	(10.4-13.2)
35-44	15%	(7.9-21.5)	11%	(9.2-13.8)
45-64	14%	(10.2-17.9)	12%	(10.0-13.5)
65+	13%	(9.9-15.2)	13%	(9.8-15.6)
Men	25%	(19.3-30.9)	18%	(15.9-20.8)
35-44	29%	(12.9-44.8)	18%	(13.7-22.3)
45-64	27%	(17.6-35.6)	19%	(15.6-21.7)
65+	22%	(15.9-29.0)	18%	(14.5-22.4)
Women	6%	(5.0-7.9)	5%	(4.0-6.6)
35-44	3%	(0.1-6.6)	5%	(3.4-7.5)
45-64	6%	(4.2-8.6)	4%	(2.9-5.3)
65+	7%	(5.1-9.2)	7%	(4.2-10.2)
Renal Impairment (Serum Creatinine Levels Above ULN, 1.5 mg/dL)				
All	37%	(31.6-41.5)	27%	(23.8-30.3)
35-44	18%	(11.1-25.0)	19%	(14.9-22.7)
45-64	28%	(22.3-33.2)	27%	(23.2-30.5)
65+	50%	(42.1-57.2)	46%	(39.1-53.5)
Men	38%	(31.3-44.1)	29%	(25.8-32.8)
35-44	24%	(9.0-38.3)	22%	(16.6-26.6)
45-64	26%	(18.0-35.0)	29%	(24.5-33.8)
65+	53%	(42.8-63.4)	47%	(39.2-54.5)
Women	36%	(30.3-41.5)	25%	(21.1-28.5)
35-44	14%	(8.0-19.1)	16%	(12.5-19.8)
45-64	28%	(21.6-35.3)	24%	(19.6-28.9)
65+	48%	(39.2-56.4)	46%	(37.0-54.4)
Renal Failure (Serum Creatinine Levels 2x ULN ≥3.0 mg/dL)				
All	0.8%	(0.4-1.3)	0.3%	(0.2-0.4)
35-44	0.0%	(0.0-0.0)	0.1%	(0.0-0.1)
45-64	0.2%	(0.0-0.5)	0.1%	(0.0-0.2)
65+	1.6%	(0.6-2.6)	1.0%	(0.5-1.4)
Men	1.0%	(0.2-1.8)	0.2%	(0.1-0.4)
35-44	0.0%	(0.0-0.0)	0.0%	(0.0-0.1)
45-64	0.3%	(-0.1-0.7)	0.2%	(0.0-0.3)
65+	2.0%	(0.1-3.9)	0.9%	(0.2-1.5)
Women	0.8%	(0.2-1.3)	0.3%	(0.1-0.4)
35-44	0.0%	(0.0-0.0)	0.1%	(0.1-0.1)
45-64	0.2%	(-0.1-0.5)	0.1%	(0.0-0.1)
65+	1.4%	(0.3-2.6)	1.0%	(0.2-1.9)

CVD indicates cardiovascular disease; HDL, high-density lipoprotein; ULN, upper limit of normal.

and somewhat contradictory. A prevalence rate of 32% was estimated in OA patients, which is higher than the 23% rate estimated for the general population. The comparatively lower prevalence of cardio-protective HDL cholesterol found in this

study is consistent with what has been reported in other studies.^{9,30-34}

Potential limitations of this study bear mention. First, it should be noted that due to the complex sampling design of NHANES III, extreme variability in the weights has the potential to result in reduced reliability of the estimates. However, the NHANES III sample was designed to minimize the variability in the weights through measures such as weight trimming. Although unlikely, extreme observations in conjunction with large weights may have resulted in extremely influential observations dominating the analyses.²⁷ Data from the NHANES surveys are considered by health services researchers to be among the most suitable-to-task for purposes of generating national estimates of disease incidence and prevalence. Nonetheless, because NHANES III is based on surveys of the civilian noninstitutionalized population, which represents 98% of the total US population, certain groups (eg, the institutionalized elderly) were excluded.³ Although the NHANES sampling methodology accounts for factors such as this, estimates of disease and risk factor prevalence presented in this article could differ somewhat from true prevalence.

Identification of comorbid medical conditions in NHANES III is derived mainly from patient self-report rather than from physical examination. Moreover, the self-reported data are confirmed by physicians only in certain circumstances. The validity of using self-reports of arthritic conditions to estimate true prevalence of OA is unknown, but studies conducted in other disease areas suggest that self-reported measures selected from NHANES can be quite reliable. The sensitivity and specificity of self-reported hypertension in NHANES III has been assessed,³⁵ and the validity of using NHANES data in this fashion for surveillance of hypertension trends in the US population is well established. Also, self-reports of an arthritis diagnosis derived from NHANES data have been used to examine the association between arthritis incidence and use of estrogen replacement therapy, body mass index,

and weight change,^{36,37} and to explore associations between arthritis diagnosis, educational attainment, and mortality.³⁸⁻⁴⁰

Because many people with arthritis may not consult a physician for their condition, they consequently may not be able to affirmatively answer the NHANES question regarding whether a doctor has told them they have arthritis. Furthermore, the possibility of faulty recall or other ascertainment bias among NHANES III participants cannot be ruled out. Patients whose health histories span many years may omit less serious health conditions, misplace dates of occurrence, or incorrectly remember the names of health conditions that were diagnosed several months or years in the past. Certainly, poor communication or a lack of understanding of medical terminology could be detrimental factors. Patients mistaking "rheumatism" for "rheumatoid arthritis" could be one example. A related concern is that the terms used to name or describe a given health condition vary among people of different language, cultural, social, or educational backgrounds. Compounding this problem is the fact that NHANES uses a checklist to collect information on chronic conditions and does not include definitions of the terms or lists of related symptoms to provide a consistent definition across subjects. However, NHANES has a deserved reputation for its clear, unambiguous diagnostic criteria and wording on its questionnaires. Although the self-reported information regarding arthritis in NHANES III has not been systematically validated, NHANES patient data has been used to ascertain prevalence of chronic disease, including arthritis, with wide acceptance since the 1970s. Self-reported rheumatoid arthritis was excluded from this analysis because it is unlikely that patients would be able to reliably report this diagnosis for all the reasons listed above.

Finally, one of the major limitations of this study was the relatively limited number of CVD risk factors that could be estimated using the NHANES III database. Interestingly, McEntegart and colleagues⁹ and Wällberg-Jonsson and colleagues^{18,41}

in their studies of RA patients identified significant correlations between RA and several thrombotic predictors of CVD that we were not able to derive from NHANES III data, including fibrinogen, von Willebrand factor, plasminogen activator inhibitor 1, tissue plasminogen activator antigen, and fibrin D-dimer. Current thinking is that inflammatory factors that promote atherogenesis and thrombogenesis may play important roles in the development of CVD in arthritis patients, particularly those with RA. Had it been possible, estimation of prevalence rates for these potential risk factors would have been worthwhile.

...CONCLUSION...

National survey data suggests, on average, US adults with OA have a high prevalence of CVD risk factors, which is higher than that of a nonarthritic population. These differences are likely to be due to the different age and gender distributions between an arthritic and nonarthritic population. Prevalence estimates, such as the ones reported here, are not conclusive evidence that OA increases the likelihood of developing CVD risk factors or CVD. If anything, they provide further evidence that CVD and arthritis may represent separate end points of a similar pathological process.^{42,43} While the importance of CVD risk factor reduction in all people is obvious, these prevalence estimates demonstrate that from a practical clinical perspective, modifiable CVD risk factors need to be aggressively managed in the arthritic population. It is important to be aware of the higher prevalence of CVD risk factors in the arthritic population when selecting from the many treatment options available today.

...REFERENCES...

1. Centers for Disease Control and Prevention (CDC). Targeting arthritis: public health takes action. Available at: <http://www.cdc.gov/nccdphp/art-aag.atm>. Accessed February 13, 2002.
2. Centers for Disease Control and Prevention (CDC). Impact of arthritis and other rheumatic conditions of the health care system—United States, 1997. *Morb Mortal Wkly Rep*. 1999;48:349-353.

3. Centers for Disease Control and Prevention (CDC). Prevalence of arthritis—United States, 1997. *MMWR Morb Mortal Wkly Rep*. 2001;50:334-336.
4. Lawrence RC, Helmick CG, Arnett FC. Estimates of the prevalence of arthritis and selected musculoskeletal disorders in the United States. *Arthritis Rheum*. 1999;42:778-799. Comment in *Arthritis Rheum*. 1999;1942:1396.
5. Centers for Disease Control and Prevention (CDC). *National Arthritis Action Plan: a public health strategy*. Atlanta, Ga: Arthritis Foundation, Association of State and Territorial Health Officials; 1999.
6. Gabriel SE, Crowson CS, O'Fallon WM. Comorbidity in arthritis. *J Rheumatol*. 1999;26:2475-2479.
7. Wällberg-Jonsson S, Johansson H, Öhman M-L, Rantapää-Dahlqvist S. Cardiovascular morbidity and mortality in patients with seropositive rheumatoid arthritis in Northern Sweden. *J Rheumatol*. 1997;24:445-451.
8. Mutru O, Laakso M, Isomäki H, Koota K. Cardiovascular mortality in patients with rheumatoid arthritis. *Cardiology*. 1989;76:71-77.
9. McEntegart A, Capell HA, Czeran D, Rumley A, Woodward M, Lowe GD. Cardiovascular risk factors, including thrombotic variables, in a population with rheumatoid arthritis. *Rheumatology*. 2001;40:640-644.
10. Gabriel SE. The epidemiology of rheumatoid arthritis. *Rheum Dis Clin North Am*. 2001;27:269-281.
11. Cerhan JR, Wallace RB, el-Khoury GY, Moore TE, Long CR. Decreased survival with increasing prevalence of full-body, radiographically defined osteoarthritis in women. *Am J Epidemiol*. 1995;141:225-234.
12. Myllykangas-Luosujärvi R, Aho K, Kautiainen H, Isomäki H. Cardiovascular mortality in women with rheumatoid arthritis. *J Rheumatol*. 1995;22:1065-1067.
13. Wolfe F, Mitchell DM, Sibley JT, et al. The mortality of rheumatoid arthritis. *Arthritis Rheum*. 1994;37:481-494.
14. Mitchell DM, Spitz PW, Young DY. Survival, prognosis, and causes of death in rheumatoid arthritis. *Arthritis Rheum*. 1986;29:706-714.
15. Mutru O, Laakso M, Isomäki H. Ten year mortality and causes of death in patients with rheumatoid arthritis. *Br Med J*. 1985;290:1797-1799.
16. Prior P, Symmons DPM, Scott DL, Brown R, Hawkins CF. Cause of death in rheumatoid arthritis. *Br J Rheumatol*. 1984;23:92-99.
17. Vandenbroucke JP, Hazevoet HM, Cats A. Survival and cause of death in rheumatoid arthritis: a 25-year prospective follow-up. *J Rheumatol*. 1984;11:158-161.
18. Wällberg-Jonsson S, Johansson H, Öhman M-L, Rantapää-Dahlqvist S. Extent of inflammation predicts cardiovascular disease and overall mortality in seropositive rheumatoid arthritis: a retrospective cohort study from disease onset. *J Rheumatol*. 1999;26:2562-2571.
19. Philbin EF, Groff GD, Ries MD, Miller TE. Cardiovascular fitness and health in patients with end-stage osteoarthritis. *Arthritis Rheum*. 1995;38:799-805.
20. Hernanz A, Plaza A, Martín-Mola E, De Miguel E. Increased plasma levels of homocysteine and other thiol compounds in rheumatoid arthritis women. *Clin Biochem*. 1999;32:65-70.
21. Maxwell SRJ, Moots RJ, Kendall MJ. Corticosteroids: do they damage the cardiovascular system? *Postgrad Med J*. 1994;70:863-870.
22. Nashell DJ. Is atherosclerosis a complication of long-term corticosteroid treatment? *Am J Med*. 1986;80:925-929.
23. Dunkin MA. Getting to the heart of the matter. *Arthritis Today*. November-December 2000. Available at: http://www.arthritis.org/resources/arthritis-today/2000_archives/2000_11_12_heart.asp. Accessed November 27, 2001.
24. Landewé RB, van den Borne BE, Breedveld FC, Dijkmans BA. Methotrexate effects in patients with rheumatoid arthritis with cardiovascular comorbidity. *Lancet*. 2000;355:1616-1617.
25. National Center for Health Statistics. *National Health and Nutrition Examination Survey, III 1988-94*. Revised October 1997. Atlanta, Ga: Centers for Disease Control and Prevention, US Department of Health and Human Services; 1997. SETS Version 1.22a.
26. National Center for Health Statistics. *Analytic and Reporting Guidelines: The Third National Health and Nutrition Examination Survey, NHANES III (1988-94)*. Atlanta, Ga: Centers for Disease Control and Prevention; October 1996.
27. Mohadjer L, Montaquilla J, Waksberg J. *National Health and Nutrition Examination Survey III: Weighting and Estimation Methodology*. Hyattsville, Md: Westat, Inc for the National Center for Health Statistics; February 1996.
28. National Institutes of Health. *The Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure*. Bethesda, Md: National Heart, Lung and Blood Institute. National High Blood Pressure Program; November 1997. NIH publication 98-4080.
29. Berkow R. *The Merck Manual of Diagnosis and Therapy*. Rahway, NJ: Merck & Co; 1992.
30. Park YB, Lee SK, Lee WK, et al. Lipid profiles in untreated patients with rheumatoid arthritis. *J Rheumatol*. 1999;26:1701-1704.
31. Philbin EF, Ries MD, Groff GD, Sheesley KA, French TS, Pearson TA. Osteoarthritis as a determinant of an adverse coronary heart disease risk profile. *J Cardiovasc Risk*. 1996;3:529-533.
32. Lazarevic MB, Vitić J, Mladenovic V, Myones BL, Skosey JL, Swedler WI. Dyslipoproteinaemia in the course of active rheumatoid arthritis. *Semin Arthritis Rheum*. 1992;22:172-180.
33. Rantapää-Dahlqvist S, Wällberg-Jonsson S, Dahlen G. Lipoprotein (a), lipids and lipoproteins in patients with rheumatoid arthritis. *Ann Rheum Dis*. 1991;50:366-368.
34. Lorber M, Aviram M, Linn S. Hypocholesterolaemia and abnormal high-density lipoprotein in rheumatoid arthritis. *Br J Rheumatol*. 1985;24:250-255.
35. Vargas CM, Burt VL, Gillum RF, Pamuk ER. Validity of self-reported hypertension in the National Health and Nutrition Examination Survey III, 1988-1991. *Prev Med*. 1997;26:678-685.
36. Sahyoun NR, Brett KM, Hochberg MC, Pamuk ER. Estrogen replacement therapy and incidence of self-reported physician-diagnosed arthritis. *Prev Med*. 1999;28:458-464.

37. Sahyoun NR, Hochberg MC, Helmick CG, Harris T, Pamuk ER. Body mass index, weight change, and incidence of self-reported physician-diagnosed arthritis among women. *Am J Public Health*. 1999;89:391-394.
38. Leigh JP, Fries JF. Correlations between education and arthritis in the 1971-1975 NHANES I. *Soc Sci Med*. 1994;38:575-583.
39. Leigh JP, Fries JF. Arthritis and mortality in the epidemiological follow-up to the National Health and Nutrition Examination Survey I. *NY Acad Med Bull*. 1994;71:69-86.
40. Hannan MT, Anderson JJ, Pincus T, Felson DT. Educational attainment and osteoarthritis: differential associations with radiographic changes and symptom reporting. *J Clin Epidemiol*. 1992;45:139-147.
41. Wällberg-Jonsson S, Cederfelt M, Rantapää-Dahlqvist S. Hemostatic factors and cardiovascular disease in active rheumatoid arthritis: an 8 year followup study. *J Rheumatol*. 2000;27:71-75.
42. Dessein PH, Stanwix AE, Moomal Z. Rheumatoid arthritis and cardiovascular disease may share similar risk factors: letter to the editor. *Rheumatology*. 2001;40:703-704.
43. Symmons D, Harrison B. Rheumatoid arthritis and cardiovascular disease may share similar risk factors: reply. *Rheumatology*. 2001;40:704.

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